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PROGNOSTIC SIGNIFICANCE OF NONINDUCIBILITY DURING BASELINE ELECTROPHYSIOLOGY STUDY VS. NONINDUCIBILITY DURING SERIAL DRUG TESTING IN CARDIAC ARREST SURVIVORS.

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It has been recommended that cardiac arrest survivors who are noninducible during baseline electrophysiologic testing (NI-B) should be considered for the implantable defibrillator. Similar recommendations are not made for cardiac arrest survivors who are inducible at baseline and who subsequently become noninducible during serial drug testing (NI-D). We compared the actuarial incidence of recurrent arrhythmias in cardiac arrest survivors who were NI-B with those who were NI-D. We also report the actuarial incidence of recurrent arrhythmias in pts who were inducible at baseline and did not respond to serial drug testing (I-NR). Of 174 cardiac arrest survivors, 66 were NI-B, 48 were NI-D, and 60 were I-NR. Fourteen pts in NI-B and 32 in I-NR received the implantable defibrillator. Recurrent arrhythmias were defined as either sudden death, or cardiac arrest, or a shock from the implantable defibrillator preceded by presyncope or syncope. The actuarial incidence of recurrent arrhythmias (± S.E.) is shown:

	1 yr	2 yr	3 yr
NI-B	5±3	18±6	22±7
NI-D	5±3	12±6	18±8
I-NR	30±6	39±7	46±8

NI-B vs. NI-D, no significant difference
NI-B vs. I-NR, $p<.02$ NI-D vs. I-NR, $p<.01$

CONCLUSION: In cardiac arrest survivors, pts who are noninducible during serial drug testing have a similar prognosis to pts who are noninducible at baseline. If additional nonpharmacologic therapy is to be considered in one group, it should be considered in both groups.

LIMITED VALUE OF PROGRAMMED ELECTRICAL STIMULATION IN PATIENTS WITH HYPERTROPHIC CARDIOMYOPATHY - EXPERIENCE IN 71 PATIENTS

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To test for pts with hypertrophic cardiomyopathy (HC) whether programmed electrical stimulation (PES) is useful in identifying pts at risk of sudden death, we prospectively collected PES and clinical data from 71 consecutive pts and related the results to a mean follow-up of 46 (range 6 to 80) months. 4 pts (group A) had a history of cardiac arrest due to documented ventricular tachycardia (VT)/ventricular fibrillation (VF). 12 pts (group B) had a history of syncope and 55 pts (group C) were uneventful with regard to syncope/cardiac arrest. Group A, B and C pts did not differ in sex, age, NYHA functional class, obstructive vs. non-obstructive type, or 12-lead and 72-h Holter ECG. All pts were off drugs during PES. PES included atrial as well as right and left ventricular stimulation with a maximum of 2 extrastimuli, during sinus rhythm and at 3 basic cycle lengths.

Results:

	Group A	Group B	Group C
non sustained (ns) VT*	1/4	2/12	7/55
sustained (s) VT**	1/4	1/12	6/55

(* ≥ 3 ventricular beats ≤ 30 sec, **VT ≥ 30sec or VF). During the follow-up period, 9 pts received amiodarone (3 Group A pts, 6 Group B pts) whereas the remaining pts were on verapamil or β-blocker therapy (7 pts). 1 pt (group C, PES: nsVT) died suddenly after conversion from verapamil to amiodarone, 1 pt (group A, on amiodarone, PES: sVT) had cardiac arrest after 3.5 years and received an automatic implantable cardioverter/defibrillator, and 1 pt (group B, on verapamil, PES: nsVT) developed recurrent syncope. No syncope or cardiac arrest occurred in the other pts.

Conclusions: 1. PES with a maximum of 2 extrastimuli does not distinguish between HC pts with or without cardiac arrest and syncope. 2. Amiodarone may not prevent cardiac arrest. 3. The low incidence of cardiac arrest in patients without a history of cardiac arrest or syncope (1 cardiac arrest among 55 pts during a mean follow-up of 4 years) confirms the benign clinical course recently reported by others.

Prognostic value of induced sustained ventricular tachycardia after myocardial infarction.

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We prospectively performed programmed electrical stimulation (PES) (single and double extrastimuli during sinus rhythm and ventricular pacing at 100 and 150 beats/min, at 2 right ventricle sites) in 211 patients (pts) without history of sustained ventricular tachycardia (SVT) one to three months (1.3 ± 0.4) after myocardial infarction (MI). During the follow up period (22 months ± 9), following events were analyzed : total cardiac mortality (TCM), sudden cardiac death (SDC) and occurrence of spontaneous SVT.

Results : PES induced SVT in 32 pts (group A) and was negative in 179 pts (group B). 11 pts were lost to follow up : 2 in group A, 9 in group B. Results are expressed in mean percentage :

	TCM	SCD	Spontenous SVT	
Group A	15.6	10	13.3	* p<.05
Group B	5.6*	3.5 NS	0.6**	** p<.001

We conclude that after MI, in patients without spontaneous SVT, systematic PES may be useful and defines a group with high risk of SVT, but not of SDC.

ELECTROPHYSIOLOGIC ABNORMALITIES IN PRIMARY CARDIAC AMYLOIDOSIS.

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Cardiac involvement in primary amyloidosis is associated with widespread amyloid infiltration of the atrial and ventricular myocardium and the conduction system. Electrocardiographic conduction disturbances are common but high-degree AV block is rare. We studied 6 patients (age 45-62, 4 male) with biopsy-proven primary amyloidosis and echocardiographic evidence of cardiac involvement. Two patients had syncope and postural hypotension, 1 wide-complex tachycardia and 3 no arrhythmic symptoms. ECG showed first-degree AV block in 3 and hemiblock in 5. AH interval was > 140 msec in 3 patients (180-210), and HV was abnormal in all 6 (mean 94 msec, range 75-105). No patient had infranodal Wenckebach with atrial pacing. The effective refractory period of the AV node was normal in all. Ventricular extrastimuli (up to 3) induced ventricular tachycardia in 2 patients (1 prior syncope, 1 prior wide-complex tachycardia) and 4 were non-inducible. Sudden death occurred in 1 patient without inducible VT, 2 died of heart failure (1 with inducible VT) 1 had recurrent syncope (inducible VT) and 2 have no arrhythmic symptoms. No patient developed heart block. We conclude that patients with primary cardiac amyloidosis have a wide variety of electrophysiologic abnormalities, most frequently a markedly prolonged HV interval. The high mortality from heart failure may account for the relative rarity of the development of high degree AV block.